ACUTE PHLEGMONOUS GASTRITIS DUE TO THE STREPTOCOCCUS PYOGENES.

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Acute phlegmonous gastritis is a comparatively uncommon finding on the postmortem table. Schnarrwyler,[1] in 1906, reviewed 83 cases that had been reported up to that time. Robertson,[2] in 1907, reported 2 cases of his own and reviewed the previously reported cases, which numbered 89. More recently Adams[3] has reported a case due to the pneumococcus. The literature upon this subject will be found in the papers referred to above.

The chief cause of acute phlegmonous gastritis is the streptococcus, but there are several cases in which the pneumococcus was found, and a few in which the condition was due to other bacteria.

The case here reported points to the symptom of sudden and severe epigastric pain as the salient clinical feature of the disease, and is of interest from both the pathological and bacteriological points of view.

E. B., male, aged thirty years, was admitted in the service of Dr. Lafleur at the Montreal General Hospital, April 26, 1910, complaining of shortness of breath and swelling of the legs.

Personal History. The patient was born in England, had scarlet fever and measles in childhood, gonorrhea at fifteen and a severe attack of typhoid fever in 1905. He was in the habit of taking one or two glasses of beer or whiskey each day, and smoked to excess, six to seven pounds of tobacco a month.

Family History. His father died aged forty-seven years, from cardiac disease, his mother of Bright's disease, aged twenty-eight years. One sister died of puerperal septicemia; three brothers and one sister are living and well.

Present Illness. February 23, 1910, after a hard day's work, he began to have dyspnea and severe pain in the right side and epigastric region. He was subsequently unable to work on account of the dyspnea. His appetite was poor and there were frequent attacks of vomiting. The pain in the right side and epigastrium were constant. On April 23 swelling of the legs was noted and the dyspnea became urgent (orthopnea). On admission the pulse was 136, irregular, of small volume and low tension; the respirations were rapid. The heart was enlarged, the sounds irregular, and there was an apical systolic murmur transmitted to the left axilla. There was edema of the legs and thighs, tenderness over the epigastrium, and slight muscular resistance over the upper part of the abdomen. The tongue was coated. As soon as food was taken vomiting occurred. The liver was palpable.

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[1] Archiv f. Verdauungskrank., 1906, xii, 116.

[2] Jour. Amer. Med. Assoc., December 28, 1907, xlix, 2143

[3] Lancet, January 29, 1910, clxxviii, 292.

He was discharged May 14, 1910, improved.

On June 9, 1910, the patient was readmitted. Soon after he left the hospital the general signs and symptoms which were present on his first admission recurred. The physical examination at this time showed an increase in the cardiac dulness and marked irregularity of the heart rhythm. There was evidence of fluid in the right pleural cavity. The tongue was coated and the breath foul. The abdomen was prominent, but there was no abdominal tenderness or rigidity. There was no dulness in the flanks. The liver was still palpable. The spleen was not enlarged.

The patient did not improve, although the edema disappeared. There was dyspnea, pain in the epigastrium, and over the precordium. Severe hiccough, with vomiting, developed and persisted.

On June 16, 1910, an area of erysipelas appeared on his nose and soon spread over both cheeks. The temperature was elevated and the precordial pain and dyspnea became more marked. The patient soon became delirious, restless, and very noisy. The tenderness over the epigastrium remained acute. On June 19, 1910, he became much worse, and complained of a sudden acute pain in the epigastrium. The pulse failed, and after severe dyspnea he died.

Autopsy. No. 107, 1910. June 19, 1910. Dr. Baird. E. B., aged thirty years; body length, 170 cm.; hour, 9.30 P.M. Service of Dr. Lafleur.

The body is that of a well-developed and well-nourished white male adult. Rigor mortis is general and well marked. There is lividity of the dependent parts and cyanosis of the face, ears, neck, and tissues beneath the finger nails. The pupils are unequal, the right 9 mm.; left, 6 mm. Slight edema over the shins and ankles. There is a dusky red, fairly sharply outlined area over the bridge of the nose and cheeks, throughout which the tissues are thickened, puckered, and of a leathery consistency.

Peritoneal Cavity. The subcutaneous fat is normal. The abdominal muscles are firm, dark red, and well developed. The peritoneum is covered with a viscid exudate, and its bloodvessels are deeply injected. In the pelvis and recesses of the peritoneum there are over 300 c.c. of thick, yellowish fluid, smears from which show Gram positive cocci in short and long chains, There is a yellowish exudate on the under surface of the diaphragm and upper surfaces of the liver and spleen. The stomach is greatly distended, dark bluish red in color; its lower border extending almost to the level of the umbilicus. The intestines are free from obstruction. There are a few small areas of necrosis in the fat about the cardiac end of the stomach. The subperitoneal and retroperitoneal tissues in the region of the left adrenal gland are edematous. The tissues about the left crus of the diaphragm are thickened and moist. On incision purulent material escapes. The mesenteric lymph nodes are enlarged, pale, and soft. The diaphragm reaches to the fifth rib on the right side and the fifth interspace on the left.

Pleural Cavities. There are no adhesions. The bloodvessels of the right pleura are deeply injected and the pleural cavity on this side contains 350 c.c. of turbid yellow fluid, smears from which shwo Gram positive cocci in short and long chains. The bloodvessels of the left pleura are less injected than those of the right and the cavity itself contains very little turbid fluid. Smears from this fluid also show Gram positive cocci in short and long chains.

The pericardial cavity is enlarged, its wall is of normal thickness. The serosa is smooth, moist, and glistening. There are 120 c.c. of clear yellow fluid present, smears from which show no organisms.

Heart. Weight, 500 grams. It measures 17 by 12.5 cm. in its longest diameters. The epicardium is generally smooth, and its bloodvessels moderately injected. Shining through the epicardium of the left ventricle are two round, elevated, light yellow, edematous areas each about 1 cm. in diameter. Smears from these show Gram positive cocci in short and long chains. The chambers of the heart are widely dilated, particularly the right ventricle, and contain blood-stained serum and postmortem clot. The myocardium is of normal consistence; that of the left ventricle is slightly thickened. The endocardium is normal. The valves are normal, except the posterior aortic cusps, which are sclerosed, much thickened, and show fenestration of their edges. The coronaries are normal. T. V., 15; P. V., 9.5; M. V., 10; A. V., 9; L. V., 2; R. V., 0.7.

Lungs. These organs are voluminous and dark red. On the posterior surface of the right lower lobe there is a round, elevated, firm, very dark red area 2 cm. in diameter. Section of the lobes shows marked congestion, and dark, frothy, blood-stained serum exudes on slight pressure. The dark red area noted above is of a much deeper red, firmer than, and is sharply separated from, the surrounding lung tissue. This area extends from the pleural surface into the lung tissue in an irregular wedge-shape.

Spleen. Weight, 310 grams, dark red. The capsule is covered with a viscid, yellowish exudate. On section the surfaces are dark red, soft, and moist, and pulp comes away freely on scraping. The Malpighian bodies are just visible.

Gastro-intestinal Tract; Stomach. This organ is markedly dilated. The bloodvessels beneath the peritoneal surface are injected. The stomach contains thin, yellowish, turbid liquid. The mucosa is generally a deep red, has a velvety appearance, and is covered with a thin tenacious layer of yellowish mucoid material. Tension shows that the deep red color is not uniform but occurs in small areas 2 to 4 mm. in diameter, separated by narrow pale bands. There are no ulcers. The wall of the stomach for a distance of 15 to 18 cm. from the esophageal orifice varies in thickness from 4 to 8 mm. On section this thickening is seen to be due to puriform infiltration of the submucosa which exudes drops of thin, yellowish pus, smears from which show leukocytes and chains of Gram positive cocci. The esophagus appears normal. The mucosa and coats of the duodenum, jejunum, and ileum are normal. The intestines contain fecal material; the mucosa in general has a pink cast.

Pancreas. Appears normal.

Liver. Weight, 1930 grams. The surface has a mottled, yellowish-red color, and near the left border of the posterior surface of the left lobe there is a deeply injected area. The edges are sharp. On section the surfaces have a greasy appearance and are of a mottled, yellowish-red color. The mottling is due to light yellow areas, varying from 1 to 4 mm. in diameter, which are separated by deep red intercommunicating bands. The liver tissue is softer than normal.

The gall-bladder is much distended and its walls are thin. Apparently there is no obstruction to the bile passages, for on exposure of the papilla of Vater bile can readily be forced out in a large stream and a probe easily passes into the common and hepatic ducts. On opening the gall-bladder two moderate sized faceted calculi are found.

Kidneys. Weight, 450 grams; enlarged, dark red, and smooth. The capsules strip readily, leaving smooth surfaces. The cortices are pale and of normal width. There is no edema. The pyramids are well defined; the pelves and ureters normal.

Adrenals. The right appears normal; the left is slightly enlarged and very deeply injected.

Bladder and Genital Organs. Normal.

Aorta. There is no sclerosis. The intima is of a deep red color throughout, appearing as if stained with red ink.

Organs of the Neck. Normal.

Brain. Weight, 1425 grams, and is normal.

Middle Ears, Nasal Sinuses, and Bone Marrow. Normal.

Anatomical Diagnosis. Acute phlegmonous gastritis; acute pleuritis; empyema; acute pericarditis; acute general peritonitis; erysipelas; central necrosis of the liver; septic spleen; cellulitis of the left crus of the diaphragm; necroses in the fat tissue about the cardia of the stomach; congestion and infarct of the lungs; cardiac dilatation and hypertrophy.

Sharlach R. Heart, spleen, kidney, no fat. Liver, cells about central veins contain fine droplets of fat.

Bacteriological Report. Cultures were made from the heart's blood and the walls of the stomach. From both of these sources a pure culture of Streptococcus pyogenes was obtained.

Microscopic Examination. *Heart.* The myocardium appears normal except that between the muscle fibers, near the epicardial surface, there are a few groups of cocci. There is no inflammatory reaction about them. The capillaries of the epicardium are deeply injected. The endocardium is normal.

Lung. Section 1: Very marked injection throughout. The alveoli are generally normal. Situated immediately beneath the pleura there is a sharply outlined area throughout which the bloodvessels and alveoli are widely distended with blood and contain a small amount of fibrin. The walls of the alveoli within this area are not distinguishable as such and are represented by faint eosin-staining bands except those just beneath the pleura, where they are better preserved. Section 2: The alveoli contain desquamated epithelium and large cells phagocytic to pigment.

Spleen. The Malpighian bodies are normal. The pulp contains a great deal of blood, in some places obscuring the lymphoid elements. There are several groups of cocci in the sinuses. The capsule is infiltrated with lymphoid and plasma cells. The arteries show some hyaline change.

Liver. The bloodvessels in the portal tissue are distended with blood. The liver cells occur as single isolated cells and short columns and groups of cells. The latter are in the peripheral portions of the lobules, are sharply separated from an inner hemorrhagic zone, and they stain deeply with eosin. There are but few liver cells recognizable in the midzonal and central portions of the lobules. They are irregularly distributed, stain poorly, generally contain vacuoles, and show varying stages of degeneration. The anatomical situation of the columns of liver cells in the inner two-thirds of the lobule is represented by poorly defined intercommunicating bands of eosin-stained material composed of a small amount of fibrin, finely granular eosin-staining material, an occasional poorly staining liver cell, a few polymorphonuclear leukocytes, numerous red blood corpuscles, cocci and endothelial leukocytes filled with yellowish pigment. The sinusoids at the periphery of the lobules are easily distinguished and contain coagulated serum and blood cells, but throughout the inner two-thirds of the lobule the sinusoids, as a rule, cannot be made out. There is no increase of connective tissue. The capsule contains a few lymphoid and plasma cells and polymorphonuclear leukocytes.

Pancreas. Normal.

Kidney. Marked injection of the bloodvessels. The epithelium of the convoluted tubules is swollen, granular, projects irregularly into the lumina, and stains poorly. The epithelium of the collecting tubules is for the most part desquamated. The glomeruli are injected, and show some hyaline change in the walls of their capillaries. Capsule normal.

Adrenal. Marked injection of its bloodvessels. There are a few lymphoid and plasma cells, and groups of cocci in the medulla.

Stomach. The following description is based upon a number of sections taken from the cardiac half of the stomach and stained by several methods.

The bloovessels are everywhere distended with blood. The mucosa, submucosa, and muscular coats show an infiltration with cells of acute inflammation. The mucosa for the most part shows only a slight degree of cellular infiltration. The lumen of some of the glands are distended with polymorphonuclear leukocytes, and in some places there are small areas of necrosis, situated both in the superficial and deep layers of the musoca.

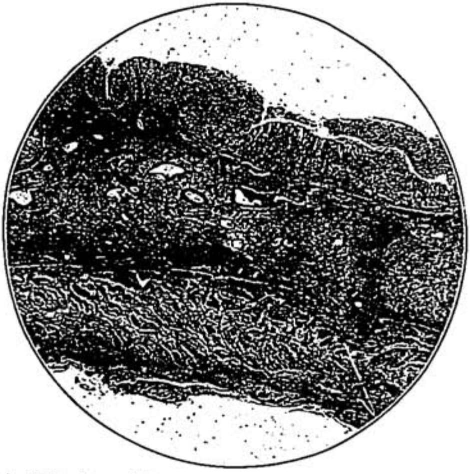

FIG. 1.—Section of stomach (low power). Note the thickened, infiltrated submucosa. The dark area to the right is shown in Fig. 2.

Between the tissues of the muscularis mucosa there are a few cells similar to those seen in the submucosa. The submucosa shows extensive necrosis, and is greatly thickened by polymorphonuclear leukocytes, small lymphocytes, red blood corpuscles, and coagulated serum. Polymorphonuclear leukocytes predominate and occur diffusely distributed through the tissue, and in small compact areas that are due to lymphatics which are distended by these cells. A similar exudate is seen in the muscular coat, but the tissues of which it is composed are not separated as much as those of the submucosa.

There are numerous groups of cocci in the stomach wall.

A section through the esophagus at the cardiac opening of the stomach shows the submucous and muscular coats infiltrated with exudate similar to that in the stomach.

Intestine. Normal.

Lymph Node. (From the gastrohepatic omentum.) The sinuses are distended with blood. There are numerous polymorphonuclear leukocytes in the pulp and sinuses, and in the sinuses there are also many endothelial leukocytes, phagocytic to cells.

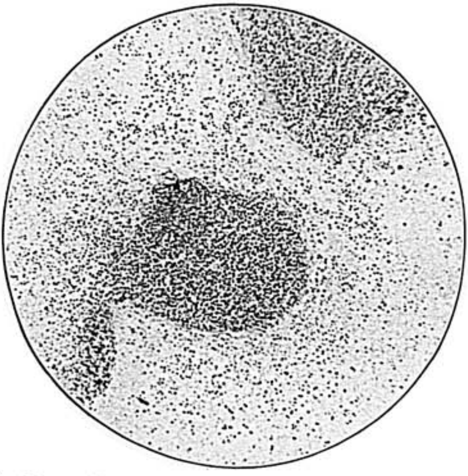

Fig. 2.—Submucosa (high power), showing lymphatics distended with polymorphonuclear leukocytes.

Phlegmonous gastritis may follow lesions in the stomach itself, or it may be secondary to an acute infection elsewhere in the body, or part of a general septicemia. It is often difficult or even impossible to determine the primary lesion. This is especially true when the stomach is but one of several organs involved and the duration of the lesions in these organs is apparently the same. Even where no ulcer is demonstrable, the stomach may be considered as the primary source of infection if this organ alone is involved, or if the acute lesions associated with it can be demonstrated to be secondary to it.

In the case reported here where there was erysipelas, acute pleuritis, acute pericarditis, acute peritonitis, and general septicemia, it is most probable that the erysipelas was primary, and the other lesions were a part of a general bacteriemia. The following may be suggested in favor of such a view: (1) Streptococcus is the common cause of erysipelas. (2) The phlegmonous gastritis in this case developed three days after erysipelas of the face. (3) There was a general bacteriemia due to the streptococcus. (4) This organism was recovered in pure culture from the stomach wall. (5) The acute lesions elsewhere in the body were apparently of the same duration as the one in the stomach, and from them the streptococcus was recovered.

In conclusion I wish to thank Dr. S. Burt. Wolbach and Dr. Lawrence J. Rhea for assistance in the preparation of this paper.

CARDIAC MURMURS DURING ATTACKS OF BILIARY COLIC.[1]

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A few years ago I called attention in a short paper[2] to the development of heart murmurs during attacks of biliary colic. Some time later, Dr. Babcock,[3] of Chicago, published an exhaustive article on the relation of the heart to diseases of the biliary tract, and within the past few months Dr. William J. Mayo,[4] in a brief but masterly discussion of diseases of the gall-bladder, made reference to concomitant heart affections. Aside from these, the literature is meager, and yet the subject has considerable practical importance. It is not my purpose to envisage the relation of the heart to the biliary tract in its entirety, but to recur briefly to the perplexing murmurs, of which, since my first paper, I have seen several additional examples. Among 56 cases of gallstone disease from private and consulting practice, 6, or 10.7 per cent., showed cardiac murmurs. This percentage does not express the real frequency of the murmur, as not all of the 56 cases were seen during or

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[1] Read at the Fourteenth Annual Meeting of the American Gastro-enterological Association, Philadelphia, April 19, 1911.

[2] The Development of Cardiac Murmurs during Attacks of Biliary Colic, Jour. Amer. Med. Assoc., May 11, 1907.

[3] Chronic Cholecystitis as a Cause of Myocardial Incompetence, Trans. Assoc. Amer. Physicians, xxiv, 43.

[4] Jour. Amer. Med. Assoc., April 8, 1911.